





A CASE OF PULMONARY EMBOLISM,  
WITH NOTES BEARING GENERALLY ON THE  
SUBJECT OF EMBOLI.

BY

WM. ROBERT SMITH, M.B.

*Honorary Medical Officer to the Hospital for Sick Children, Sheffield.*

*(Reprinted from THE MEDICAL TIMES AND GAZETTE.)*

LONDON :

PARDON & SON, PRINTERS, PATERNOSTER ROW.

1877.

c



A CASE OF PULMONARY EMBOLISM, WITH NOTES  
BEARING GENERALLY ON THE SUBJECT OF  
EMBOLI.

*(Read before the Sheffield Medical Society.)*



IN connexion with the notes of the case which I am shortly to read, I have thought that perhaps a brief review of the subject of embolism might not be out of place; and although I have no original views to advance on this most important disease—important alike to the physician, surgeon, and accoucheur—and have consequently hesitated in bringing the subject before you, yet I do feel that if by discussion any new light can be thrown upon the pathology or treatment of a disease which is unfortunately so frequently attended with the worst results, our meeting this evening will not be devoid either of interest or profit.

The existence of the morbid condition which has been designated embolism was, to a certain extent, known at a very early period in the history of our art, and has over and over again been established by well-authenticated cases and carefully made post-mortem examinations. Thus, a connexion between gangrene of a limb and plugging of the main artery, between apoplexy or sudden asphyxia and hyperinosis of the blood or the so-called polypi of the heart, has long

been recognised and looked upon as cause and effect. Yet it is to Virchow, as all know, that science is indebted for having first placed, by his many admirable researches, the theory of emboli upon a firm foundation. But before his time embolism did not pass unnoticed. Even Galen states ("De Loco Adfecto," iv., page 295) that he often met with cases of sudden death in persons labouring from cardiac affections; in such he suggested as the cause the sudden obliteration of the pulmonary arteries by bodies which were subsequently called cardiac polypi. But later on the importance of embolism was more fully recognised; for Vesalius, in the sixteenth century, relates ("De Gangrena et Sphacelo," c. 4, page 775) a case of an enormous polypus in the left cardiac ventricle, weighing two pounds, and which gave to the heart the size and form of the gravid uterus. At the same time, he states that with the growth of this mass gangrene of the extremities is often combined; and that slight irregularity of the pulse is the only sign by which this condition is revealed. In short, the whole doctrine of emboli, by which I mean the plugging of vessels by foreign bodies, was first based upon the theories suggested by the supposed spontaneous formation of the so-called polypi within the vascular system. From this time much was written and said about embolism, but still no great advance seems to have been made upon the teaching of the older writers; for we find Cruveilhier, notably with many others, advocating that if any plug was found in the vascular system it was to be considered as a local product, the result of inflammation or some other process—the some other process, however, never being very distinctly defined; and thus a new variety of arteritis was taught, consisting essentially in the formation of a coagulum, the co-existence of inflamed arterial walls being considered altogether beside the point. In 1828, Alibert reported a case ("Recherches sur une Occlusion peu connue des Vaisseaux Artériels considérée comme Cause de Gangrène") of gangrene of the

extremities, and obstruction to the circulation in the left arm, and distinctly showed the resemblance of the coagula which existed here to that of certain fibrinous formations in the left cardiac auricle. Immediately after this, Victor François published a work, in which sudden arterial obstruction was taught to be caused by foreign plugs, the result of a more or less distant arteritis, and which had been carried along by the circulating fluid. These views, however, made but little headway, for Tiedemann, in his work on the "Constriction and Closing of Arteries in Disease," published in 1843, mentions that coagulations, which, like stoppers, block up the arterial canals, may be either processes of the fibrinous layers of aneurismal sacs extending more or less into the arteries; or simple depositions of fibrine upon the sharp points of earthy concretions projecting through the inner arterial coat into the canal. Here there is evidently no thought of embolism. Four years after the publication of this book, a case manifestly depending upon emboli was published in the *Gazette Médicale de Paris*, page 672, by Pioch. A patient, labouring under hypertrophy of the heart and valvular induration, had several sudden arterial obstructions occurring in the brachial, vertebral, and right and left femoral vessels. Gangrene followed in the extremities, but no post-mortem was allowed; about this time also (but I forget where) another case was published, where obstruction in several main trunks existed subsequent to rheumatism, and accompanying endocarditis, but there was no gangrene. In the same year, Professor Virchow, of Berlin, first published a paper on Arteritis, in the *Archiv für Physiologische Anatomie*, in which he questioned many of the generally received views, and clearly showed that those products which had been taught to be the result of inflammation were of embolic origin. Virchow also taught that when a clot existed in the pulmonary artery long before death, and when, subsequent to this obstruction, changes took place

in the parenchyma, such obstructions have always arisen in a part of the circulation anterior to the pulmonary arteries, either in the right side of the heart or systemic veins; he supported this view by a series of experiments introducing animal substances, etc., and produced (if you will allow me to quote his own words from page 15 of his work) "violent pneumonias, commencing with inflammatory hyperæmia, and causing the rapid deposition in the pulmonary vesicles of fibrinous exudations, which either underwent purulent metamorphosis or became gangrenous. With the advance of these changes, pleurisy was soon developed at the periphery, at first producing fibrinous, coagulable, and viscid exudations over the affected portion of the lung, but rapidly and as it progressed toward the other side of the chest, accompanied with enormous increase of hyperæmia, extravasations in the parenchyma of the pleura, and large watery hæmorrhagic exudations, with prepoudering tendency to ichorous metamorphosis in its cavity. At the affected part of the lung the pleura became gangrenous, and finally gave way, and pneumothorax set in. The whole series of phenomena was developed in not quite five days."

Shortly after this another work of great interest to the surgeon was published—viz., "Investigations on Inflammations of the Arteries in general." Physiologists had long taught that the inner coat of arteries had no bloodvessels: How then could an exudation be produced through it? Was this to be looked upon as the result of a local inflammation? or was it the local manifestation only of a generally morbid blood condition? or was it, in the third place, a solid body, which, by some means or other, had entered the circulation, and got carried on with the stream until it entered a vessel too small to allow of its passage? Arteritis certainly could not exist in the deposit of an exudation upon a free surface; abscesses had been found between the inner and middle coats of arteries, without any correspond-



ing production in the arterial cavity ; in many cases gangrene of the limbs was found co-existent with plugging of the main arterial branch. Now, in such cases was the obstruction primary, and the inflammation of the vessel a secondary matter caused by the irritating properties of the embolon ? or was the relation reversed ? or, in the third place, were the obstruction and inflammation the joint effects of a cause separate and distinct ? According to Virchow, the inner coats of arteries are not permeable to inflammatory exudations from the vasa vasorum of such vessels, this exudation being capable of coagulation ; but this is found either between the outer and middle coats, or between the middle and inner ones ; and this latter must give way before pus can be deposited in the arterial cavity. Sometimes coagula are found in an inflamed vessel when they are caused by a deposition of fibrine upon a roughened arterial wall, or upon some projection through such wall, the result of imperfect nutrition. In some cases the coagulum adheres only to one wall of the vessel, with the effect of narrowing the tube. This is not true embolism, although such a condition may give rise to embolic obstruction. Embolism may be defined as the existence of a clot in an artery without simultaneous lesion of its walls, this clot having been carried with the circulating stream from some distant spot as far as it would go, the arrest always taking place in vessels of a suddenly constricted calibre, too small to allow the solid body to pass. Such are briefly the views set forth in Virchow's work on *Arteritis*.

Since his time many able observers have written upon the subject, but their works are for the most part all based upon Virchow's theses, and are confirmatory of his observations. Time will not allow of reference to these, but I would remind you of Dr. Kirkes' conclusions from a careful study of the whole subject—viz., (1) that fibrous conerctions did separate from the heart during life ; (2) that these were arrested in

certain peripheric organs, where they caused obstruction and structural changes; and (3) that by simple admixture with the blood they might give rise to pyæmia or other blood-poisoning conditions. That part of his observations is most interesting where he explains the petechial spots with which we are all familiar as very frequently occurring in endocarditis and pyæmia, on the skin, mucous membranes, and even on the peritoneum, as being caused by capillary embolisms. Since this paper appeared in the *Lancet* of June, 1852, we have learnt to attribute many sudden attacks of hemiplegia to embolic causes; and those researches are very interesting upon brain-softening as the result of embolism, where the anæmia of the brain is clearly shown to be the cause of the softening. And yet this term "anæmia" is a bad one, for very soon the collateral vessels enlarge and give way; extravasation then takes place, giving rise to a uniform red appearance, and that condition which we know as "hæmorrhagic infarct," and red softening. It is only when the circulation is very slightly interfered with, or when it is materially affected (as, *e.g.*, by plugging of the middle cerebral), that we get white softening. Here we see that brain-softening as the result of embolism, unlike senile gangrene, is not caused by a mere deficiency of blood, but is owing to serous effusion, the consequence of hyperæmia and stasis, the remote cause being the embolus. These hæmorrhagic infarcts are now also well known to exist in the lungs, liver, kidneys, and spleen, and present subjects of the deepest interest to the pathologist.

The most frequent source of emboli is undoubtedly venous thrombi, this giving rise to it in many ways: the thrombus may become softened and disintegrated, the fragments being carried into the circulation; or the blood-clot may not for some reason or other, fill the vessel—the blood passing over it in this condition is very liable to carry away portions, or the whole, into the general circulation; or, thirdly (and this per-

haps is the most frequent way), the conical clot, which extends in a ruptured vessel as far as the first branch, is very prone to have its apex broken off by the onward rush of blood, which is always stronger at this place, owing to the obstruction forming a point which the blood can wash round, and which momentarily impedes its progress. If the embolus is found in the veins, it may pass through the right side of the heart and become impacted in the lungs; but if small enough it may traverse the pulmonary capillaries, pulmonary veins, and left heart, and cause obstruction in the renal or splenic capillaries. The pathological results of such obstruction are most interesting. First there is complete perversion of function, followed sooner or later (according to the more or less rapid formation of the collateral circulation) by a zone of intense hyperæmia around the area of tissue from which the blood-supply has been removed. Very soon, if the circulation be not re-established, the embolic area becomes distended with blood, owing to extravasation from the over-distended capillaries, the blood regurgitating from the veins. The subsequent changes depend upon the character of the embolus: if this be small, and not infective, then the mass softens, the blood changes from red to brown or yellow, and the whole contracts and becomes shrivelled, but is always surrounded by a zone of hyperæmic tissue; but if the embolus is caused by a softening and disintegration of the thrombus (say, *e.g.*, after an amputation, and when the stump looks unhealthy), then the embolus is associated with the infective properties of putrefaction and suppuration, and a number of metastatic abscesses appear in various organs as the result of impaction, and that condition which the surgeon most dreads—viz., pyæmia—exists. The point is not yet settled, I believe, whether true pus can cause capillary embolism, but there can hardly be a doubt, one would think, that if pus cells existed in such quantities as to occlude any capillary vessel, the pathological results

with which we are all familiar as supervening upon embolism would follow. There is a point of much value practically in connexion with this surgical view of the subject, worthy of note. I believe it was first suggested by Professor Burrow. He states that the frequent bandaging of limbs after amputation is the cause of many unfavourable results; the veins are compressed by this application, and after a few days, when the bandage is removed, the venous walls of course become relaxed, and the coagula no longer fill the vessels, and are liable to be washed away by the passage of the blood over them. Embolism, and not pyæmia, is really the cause of death in such cases. I well remember a case which occurred whilst I was house-surgeon, which no doubt was owing to this cause. A healthy man had his thigh amputated after a severe crush of the leg implicating the knee-joint, and a bandage was applied round the stump to control the excessive spasm of the muscles. All went on well for some time—the wound looked healthy, and the man's general condition was good; but he subsequently had one or two severe rigors and died, and after his death several abscesses were found in his lungs. This case was registered as death from pyæmia, but I believe it was entirely of an embolic origin, caused by the removal of the bandage, and consequent relaxed venous walls. This supposition, I know, some will take exception to. Still, considering the man was placed under the best hygienic conditions, that the most scrupulous care was taken as to cleanliness and the dressing of the wound—the antiseptic treatment being adopted,—and the absence of any unhealthy appearance of the stump, I think my impression not altogether an erroneous one.

To the accoucheur the frequent and sudden fatal results of embolism after parturition are too well known and cannot fail to make the subject interesting. His patient may be bright and cheerful, and he may be congratulating himself upon the very favourable progress of the case, when all at once he is

summoned only to find her a corpse. The following case will illustrate this :—

On the morning of February 24, about 6 a.m., I was called to attend a woman, aged twenty-four years, in her second confinement; for some months she had suffered severe pain in the abdomen and back, accompanied during the preceding week with much nausea and vomiting. As far as I could learn, she had been very injudicious during her pregnancy, carrying heavy weights, and generally busying herself too much about household duties. Even the night her labour commenced she had carried a heavy child of two years up a flight of stairs. I found Mrs. W. suffering severely from sharp acute pains, not of a bearing-down character, which were very irregular in their recurrence, but for the most part frequent. She had also constant vomiting. Her countenance was anxious; pulse fair volume, 82 per minute; skin cool and moist. Before I could make an examination, unfortunately, the membranes broke, and the liquor amnii escaped in large quantities, soaking the mattress, and dripping on to the floor beneath. This fact first awoke my suspicions that all was not right. Upon making a vaginal examination these were confirmed, for I found the right hand presenting, the child lying in the right oblique diameter, with its back to the mother's abdomen. The mother, however, was very sensitive, and it was with great difficulty I could satisfactorily make a diagnosis as to the child's position. I saw that any attempt to turn without administering chloroform would be entirely futile, so, in spite of the serious sickness from which the patient was suffering, I used this anæsthetic; then, introducing the left hand, I grasped the left foot, and completed the operation of version, afterwards delivering the child in the way usually adopted in breech cases. The placenta followed in due course, and the uterus became firmly contracted. Matters, however, did not go on so smoothly with the child: for fifteen minutes artificial respiration had to be kept up

before it evinced any signs of life, then by vigorously slapping the buttocks it began to cry lustily.

Twelve hours after I found Mrs. W. feeling very comfortable. She had not been sick, but had felt great nausea. Complained a little of after-pains; skin cool and moist; pulse 110. Had passed water, and the discharge was not excessive. Milk was ordered to be given in small quantities frequently, and ice was allowed.

February 25.—Has slept fairly well; expresses herself as feeling better, and complains less of after-pain; has had no recurrence of the vomiting, but still feels much nausea. Temperature  $101.4^{\circ}$ ; pulse 120, good volume. Has no abdominal tenderness; lochial discharge plentiful. The patient was ordered still to persevere with the milk and ice, and a small quantity of sago or arrowroot was likewise allowed, perfect rest in the horizontal position was enjoined, and the temperature and ventilation of the room were carefully attended to.

26th.—Has slept well; countenance cheerful; feels much better; tongue clean and moist; complains less of the nausea; bowels moved gently this morning; appetite good, the patient asking for a more substantial diet; discharge plentiful and healthy; milk abundant. Temperature  $100.6^{\circ}$ ; pulse 96. The same treatment was persevered with, beef-tea and sago-pudding being ordered in addition.

From this date the patient progressed very favourably until March 1, when I found her by no means so cheerful. She had had a slight rigor the night before, which came on after her removal to another bed, which had been done without my knowledge; had slept badly, and complained of great pain in her breasts, which were found to be swollen and hard; tongue furred, with a dryish white fur on dorsum; bowels not moved the day before. Temperature  $103.2^{\circ}$ ; pulse 126, irregular and jerky; lochial discharge abundant; no abdominal tenderness;



patient's countenance was anxious, and she expressed herself as feeling very ill and depressed. Milk diet was ordered, a gentle aperient given, a simple effervescing draught ordered three times a day, and hot flannels were applied to the breasts.

March 2.—Has slept well, and feels better this morning; tongue less furred; bowels moved freely twice; has taken her nourishment better. Temperature  $102.4^{\circ}$ ; pulse 120, irregular and rather jerky.

3rd.—Much better; slept well; tongue clean; is more cheerful; temperature  $102^{\circ}$ ; pulse 120, still distinctly irregular. At 5 p.m. I was summoned to see the patient at once, as she was much worse; upon visiting her two hours afterwards, when the message reached me, I found her dead. As far as I could learn from the husband, who was present, it appears she had raised herself in bed to use the bed-pan, and felt suddenly so ill that she laid down again, and died within a quarter of an hour with all the signs of asphyxia. The face was pale, the eyes were open, pupils widely dilated, the muscular system rigid, and hands firmly clenched.

*Autopsy, twenty-four hours after Death.*—Rigid mortis present; body well nourished. Upon opening the chest, the left lung was found collapsed, although the right was by no means over-distended. Heart: Pericardium contained a small quantity of serum; right cavities and large veins distended with dark fluid blood; left chambers empty. Trachea congested, and the left bronchus contained a little frothy fluid. Lungs: Right normal; hypostatic congestion at base. Left—upon tracing the branches of the pulmonary artery into its substance, at the site of the bifurcation of one of the main branches, an embolism, composed of clot, was found firmly wedged; it was about three-quarters of an inch in length, and rather larger round than a piece of slate-pencil. The lung-tissue was congested about the site of the embolus, but there was no distinct infarct.

Uterus firm, five inches in length, and seven-eighths of an inch in thickness; no signs of inflammation present.

The embolus in this case I believe to have been detached from the uterine sinuses at the time my patient walked from one bed to the other. If this hypothesis is correct, the embolus was probably detained in the heart by the relaxed tendinous cords which are so abundant in the right ventricle, and thus gave rise to the high temperature, and quick, jerky pulse. At any rate, I think it is clear that, if this were the case, treatment, to be of service, should then have been adopted, but unfortunately at this stage there is a marked absence of symptoms sufficiently reliable to insure a satisfactory diagnosis. I believe the rigors which my patient had were owing to the sudden appearance of a clot in her circulation, and not, as I then believed, to the swelling and tenderness of the breasts: yet why should this not have been the cause? or how is it possible to diagnose with certainty a condition of so much danger to the patient? The only symptoms I could not explain were the continued high temperature and quick pulse after the mammary trouble had subsided. I know some will urge that the embolus might have been separated directly at the time of death, but I think the absence of all inflammation of the veins and uterus goes far to negative this idea. It is with the view of learning something as to the treatment and diagnosis of such cases, from those who have had far more experience than myself, that I have ventured to trouble you, at, I fear, some length, with this subject to-night.





